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The Ventriculo-Arterial Coupling Ratio During Transient Gz Events

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Abstract

It has been shown by Westerhof et al. that the ratio τ/T (where $\tau = R_p * SAC$ and R_p is total peripheral resistance, SAC is systemic arterial compliance, and T is heart period) is approximately a constant in all mammals under resting conditions such that diastolic pressure is sufficiently high to assure adequate coronary perfusion. The aim of this study is to determine if the ratio τ/T is constant under the transient condition of a rapid onset rate +Gz acceleration. We hypothesize that the ratio is held constant by the cardiovascular control system. Four male baboons were subjected to 10 second rapid onset +Gz profiles and aortic pressure and flow were recorded. R_p and C were calculated using a 2-element windkessel model and T was determined by the inverse of heart rate. All parameters were calculated on a beat-to-beat basis. It was found that the ratio increased very little at +2 Gz and increased dramatically at +3 and +5 Gz, disproving our hypothesis. This increase was due to large increases in peripheral resistance and changes in heart rate over the course of a Gz run. The increases are presumed to represent baroreceptor reflex response to the drop in aortic pressure experienced during +Gz stress. The ratio τ/T returned to its initial resting value shortly after the +Gz stressor was removed.

Introduction

Ventriculo-arterial coupling can be defined as the maintenance of blood flow through the systemic capillary beds during systole, and the perfusion of the heart muscle during diastole. Optimal ventriculo-arterial coupling can be viewed in many ways, including maximum external work, maximum mechanical power, and maximum efficiency [1]. However, during rapid onset rate (ROR) +Gz head-to-foot acceleration, several cardiovascular control systems are activated which affect changes in several coupling parameters. Of primary interest is the regulation of pressure by baroreceptor reflexes. At the onset of a

higher G-force than normally experienced by the body, aortic pressure begins to drop quickly. This triggers a response by baroreceptor reflexes, initiating changes in total peripheral resistance ($R_p = TPR$), heart rate (HR), and systemic arterial compliance (SAC). As a result, arterial decay time ($\tau = R_p * SAC$) and heart period (T) also change. Westerhof et al. have shown that the ratio of arterial decay time to heart period (τ/T) is approximately a constant over all mammals, and may be an index of optimal ventriculo-arterial coupling [2]. It is proposed that this ratio is held constant by the cardiovascular control system during transient Gz events in order to assure optimal coupling.

Methods

Four clinically normal male baboons were used for this study. All were cared for under the provisions of the *Guide for the Care and Use of Laboratory Animals* (NIH No. 80-23). Diet consisted of Purina monkey chow biscuits and fresh fruit. The baboons were instrumented under general anesthesia as described previously [3]. A 3-Fr micromanometer-tipped pressure catheter (Millar Instruments Inc., Houston, TX) was used to measure aortic pressure (AoP) and an implanted EMF flow probe was used to measure aortic flow (AoF). Before each centrifugation, the baboons were sedated with Ketamine (5-10 mg/kg), and were placed in confinement chairs bolted to the end of the Brooks AFB centrifuge arm. A series of ROR (5 G/s) were performed ranging from 2 to 5 +Gz levels for a duration of 10s each. The physiological signals were stored on VHS tape and A/D converted for use with computer.

A two-element Windkessel model was used to estimate the vascular parameters R_p and SAC from the pressure and flow data obtained [4]. R_p and SAC were filtered using a 3-point median filter. Heart rate was calculated by the use of a cardiovascular data handling software package implemented in MATLAB™ (The Math Works Inc., Natick, MA) [4]. Statistical analysis was done using a paired two-sample student's t-test on the data.

Results

Figure 1 shows a representative plot of the normalized ratio τ/T shown with the scaled Gz signal at 2, 3, and 5 Gz plotted versus time. Figure 2 shows significant changes in the magnitude of the τ/T ratio between maximum and control values at 5 G, while the ratio at 2 G is nearly the same at both control and at maximum amplitude. There is also a statistical difference between the value of τ/T at control and at its maximum at all Gz levels. It is shown in Figure 3 that τ/T reaches a maximum more quickly than it recovers. Figure 3 also indicates that the time of recovery doesn't change between 3 and 5 Gz. The change in the ratio τ/T is caused by TPR and heart rate (Figures 4 and 5). The increase is driven primarily by TPR which increases to a higher relative magnitude than heart rate. SAC shows very little statistical difference between control and maximum values and appears to play little role in changing τ/T (Figure 6). Figure 7, 8, and 9 show the time of recovery of TPR, heart rate, and SAC, respectively. TPR and SAC show little change in recovery time over 2, 3, and 5 Gz (Figures 7 and 9). Heart rate takes longer to recover as Gz increases (Figure 8). Comparing recovery times of τ/T and the parameters that comprise it, it is shown that τ/T recovers more quickly than its components. Figures 2 through 9 show the results of the student's t-test, giving the probability that both data sets came from the same sample.

Discussion

As can be seen in Figures 1 and 2, the ventriculo-arterial coupling ratio changes over the course of a transient +Gz event. The ratio changes as the cardiovascular control system attempts to counteract the decrease in AoP that occurs during a 10 second ROR +Gz profile. It does so by increasing TPR and heart rate, thereby decreasing heart period (T). SAC shows no influence on τ/T . The increase in τ/T is driven primarily by TPR (Figure 4), which can increase to a higher relative magnitude than heart rate (Figure 5). By increasing TPR, the arterial decay time constant increases, allowing the coronary arteries to be perfused at a higher average pressure. This helps to maintain coupling necessary for coronary perfusion, but the increased TPR leads to decreased flow through the systemic capillary beds. From Figure 1, it is shown that τ/T is not truly a constant under steady state, but has some variation, especially after undergoing +Gz. We have found that τ/T reaches its maximum near the time of the offramp of the 10 second ROR +Gz profile. Recovery time is approximately the same at 3 and 5 Gz, implying that the control system may have reached saturation. In addition, the ventriculo-arterial coupling ratio recovers to its control value faster than the parameters that comprise it (Figure 3 and Figures 7-9). This may suggest that τ/T is parameter controlled by baroreceptor response.

Conclusion

We have looked at the ratio τ/T as an index of optimal ventricular arterial coupling. We found, contrary to our hypothesis, that the ventriculo-arterial coupling ratio is not constant over the course of a +Gz transient. This may indicate that optimum coupling is not realized due to inadequate coronary perfusion or lower flow through the systemic capillary beds. It may also imply that the cardiovascular control system can not respond to the rapid changes in Gz. It has also been shown that the ventriculo-arterial coupling ratio returns to its control value more quickly than its component parameters that it is comprised of. This may indicate that changes in the ventriculo-arterial coupling ratio represent responsiveness of baroreceptor mediated reflexes.

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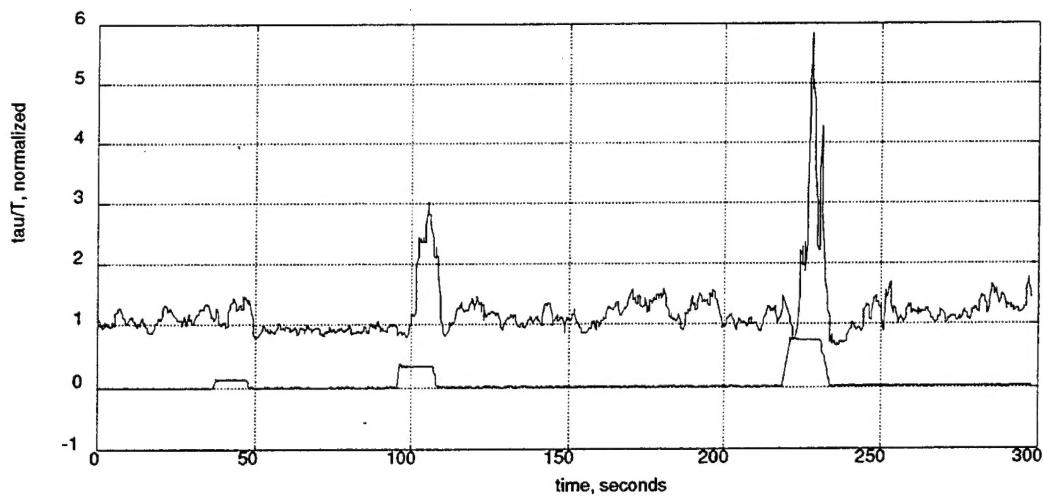


Figure 1. Representative plot of the ratio τ/T during 2, 3, and 5 Gz ROR profiles. τ/T has been normalized and Gz has been scaled. The top trace is τ/T and the bottom trace is Gz.

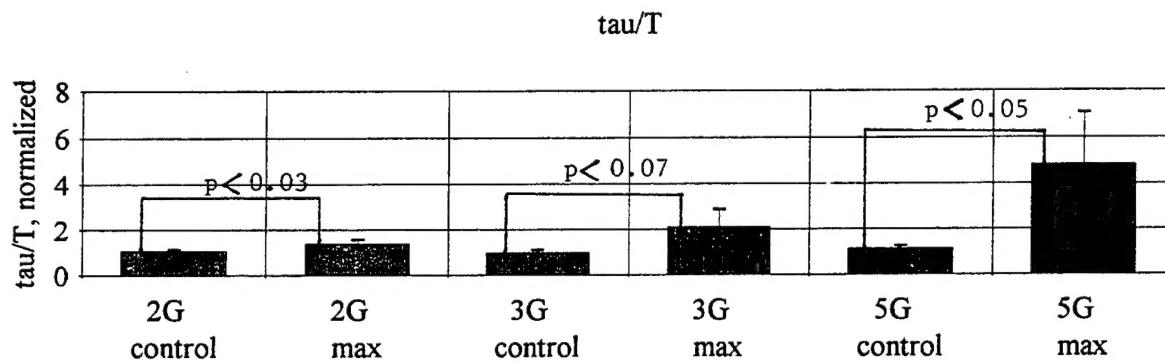


Figure 2. Mean values for τ/T at control and maximum amplitude for each +Gz level. The magnitudes were normalized to themselves at a steady state point before the +2 Gz ROR. Error bars show standard deviation.

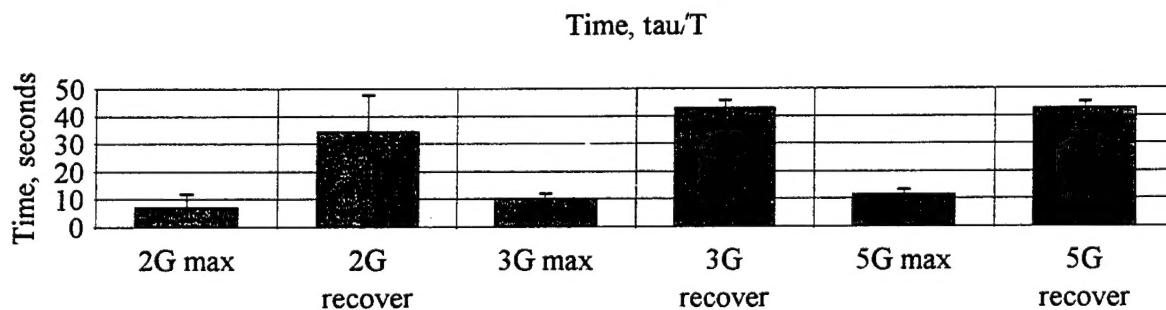


Figure 3. Mean values for the time from control to maximum response and time from maximum to recovery. Error bars represent standard deviation. $p < 0.05$ for all cases.

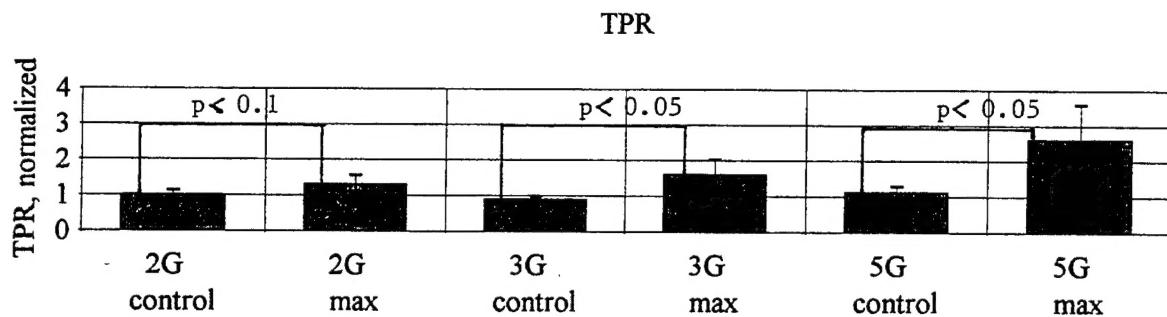


Figure 4. Mean values for TPR (R_p) at control and the point of maximum amplitude of τ/T for each +Gz level. The magnitudes were normalized to themselves at a steady state point before the +2 Gz ROR. Error bars show standard deviation.

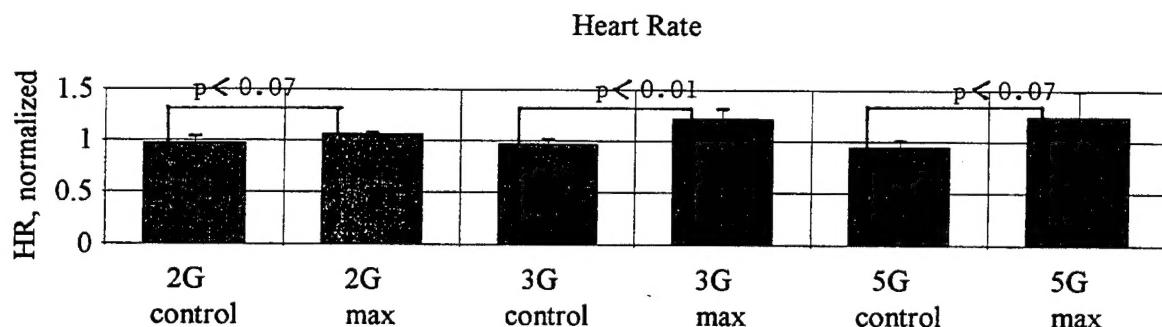


Figure 5. Mean values for $HR = 1/T$ at control and the point of maximum amplitude of τ/T for each +Gz level. The magnitudes were normalized to themselves at a steady state point before the +2 Gz ROR. Error bars show standard deviation.

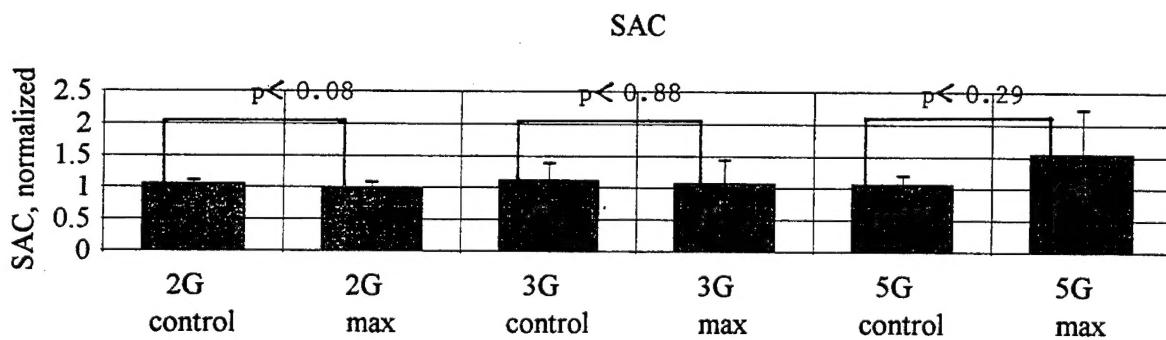


Figure 6. Mean values for SAC at control and the point of maximum amplitude of τ/T for each +Gz level. The magnitudes were normalized to themselves at a steady state point before the +2 Gz ROR. Error bars show standard deviation.

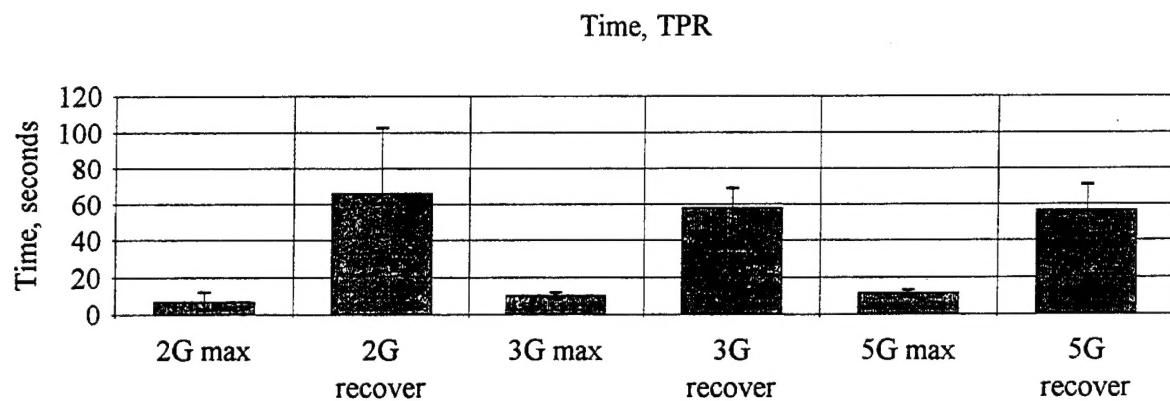


Figure 7. Mean values for the time from control to maximum τ/T and from that point to recovery of the parameter TPR. Error bars show standard deviation. $p<0.05$ for all cases

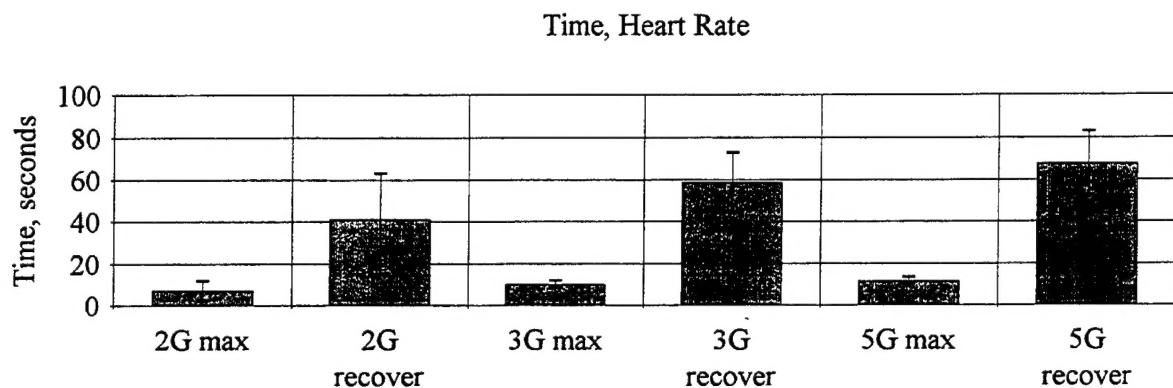


Figure 8. Mean values for the time from control to maximum τ/T and from that point to recovery of the parameter heart rate. Error bars show standard deviation. $p<0.05$ for all cases.

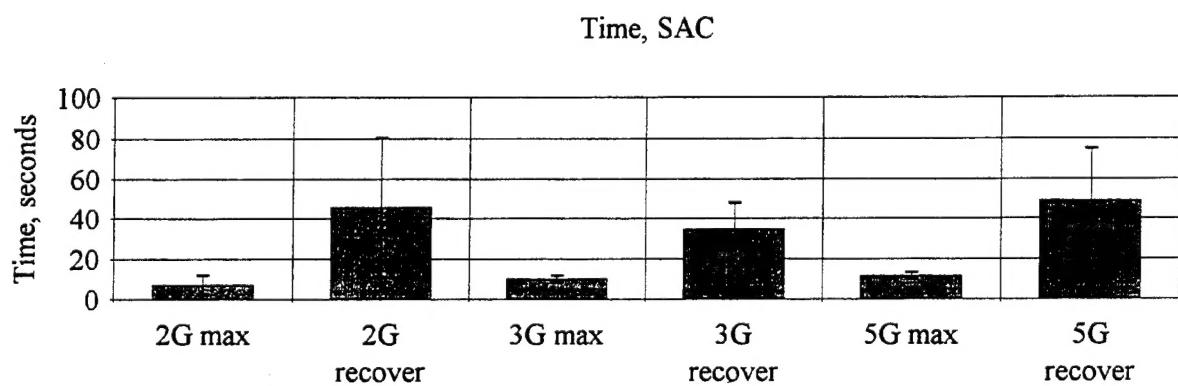


Figure 9. Mean values for the time from control to maximum τ/T and from that point to recovery of the parameter SAC. Error bars show standard deviation. $p<0.05$ for all cases.